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FILE 'HOME' ENTERED AT 13:35:03 ON 04 FEB 2004

=> file medline  
COST IN U.S. DOLLARS  
FULL ESTIMATED COST

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0.21	0.21

FILE 'MEDLINE' ENTERED AT 13:35:09 ON 04 FEB 2004

FILE LAST UPDATED: 3 FEB 2004 (20040203/UP). FILE COVERS 1958 TO DATE.

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This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> s globin promoter
      9966 GLOBIN
      9769 GLOBINS
     13089 GLOBIN
           (GLOBIN OR GLOBINS)
      97282 PROMOTER
     22659 PROMOTERS
    104410 PROMOTER
           (PROMOTER OR PROMOTERS)
L1      387 GLOBIN PROMOTER
           (GLOBIN(W) PROMOTER)
```

```
=> s l1 and locu control region
           2 LOCU
      69856 LOCUS
      69856 LOCU
           (LOCU OR LOCUS)
    1420348 CONTROL
      298212 CONTROLS
    1590601 CONTROL
           (CONTROL OR CONTROLS)
      371222 REGION
     259983 REGIONS
     547962 REGION
           (REGION OR REGIONS)
           715 LOCU CONTROL REGION
           (LOCU(W) CONTROL(W) REGION)
L2      84 L1 AND LOCU CONTROL REGION
```

```
=> s l1 and locus control region
      69856 LOCUS
       29 LOCUSES
     39571 LOCI
       7 LOCIS
     96383 LOCUS
           (LOCUS OR LOCUSES OR LOCI OR LOCIS)
    1420348 CONTROL
      298212 CONTROLS
    1590601 CONTROL
           (CONTROL OR CONTROLS)
      371222 REGION
     259983 REGIONS
     547962 REGION
           (REGION OR REGIONS)
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716 LOCUS CONTROL REGION  
(LOCUS (W) CONTROL (W) REGION)

L3 84 L1 AND LOCUS CONTROL REGION

=> d bib ab 80-84

L3 ANSWER 80 OF 84 MEDLINE on STN  
AN 93087190 MEDLINE  
DN 93087190 PubMed ID: 1454538  
TI Structure and function of the murine beta-globin locus  
**control region** 5' HS-3.  
AU Hug B A; Moon A M; Ley T J  
CS Department of Medicine, Jewish Hospital, Washington University Medical  
Center, St Louis, MO 63110.  
NC CA 49712 (NCI)  
DK 38682 (NIDDK)  
SO NUCLEIC ACIDS RESEARCH, (1992 Nov 11) 20 (21) 5771-8.  
Journal code: 0411011. ISSN: 0305-1048.  
CY ENGLAND: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
OS GENBANK-Z13985  
EM 199301  
ED Entered STN: 19930129  
Last Updated on STN: 19930129  
Entered Medline: 19930104  
AB We previously identified the murine homologue of the human beta-globin  
**Locus Control Region (LCR)** 5' HS-2. The  
lambda clone containing murine 5' HS-2 extends approximately 12 kb  
upstream from this site; here, we report the sequence of this entire  
upstream region. The murine homologue of 5' HS-3 is located approximately  
16.0 kb upstream from the mouse epsilon y-globin gene, but no region  
homologous to human 5' HS-4 was present in our clone. Using a reporter  
system consisting of a human gamma-globin promoter  
driving the neomycin phosphotransferase gene (gamma-neo), we tested murine  
LCR fragments extending from -21 to -9 kb (with respect to the epsilon  
y-globin gene cap site) for activity in classical enhancer and integration  
site assays in K562 and MEL cells. 5' HS-2 behaved as a powerful enhancer  
and increased the number of productive integration events (as measured by  
a colony assay) in both K562 and MEL cells. 5' HS-3 had no activity in  
K562 cells or in transiently transfected MEL cells, but was nearly as  
active as 5' HS-2 in the MEL cell colony assay. Two additional tests  
confirmed the identification of murine 5' HS-3: first, a DNA fragment  
containing 5' HS-3 confers copy number-dependent, integration-site  
independent inducibility on a linked beta-globin gene in the MEL cell  
environment. Secondly, a strong DNaseI hypersensitive site maps to the  
location of the 5' HS-3 functional core in chromatin derived from MEL  
cells. Collectively, these data suggest that we have identified the  
murine homologue of human 5' HS-3, and that this site is functional when  
integrated into the chromatin of MEL cells but not K562 cells. 5' HS-3 may  
therefore contain information that contributes to the development-specific  
expression of the beta-like globin genes.

L3 ANSWER 81 OF 84 MEDLINE on STN  
AN 93087174 MEDLINE  
DN 93087174 PubMed ID: 1454528  
TI The developmental regulation of the human zeta-globin gene in transgenic  
mice employing beta-galactosidase as a reporter gene.  
AU Pondel M D; Proudfoot N J; Whitelaw C; Whitelaw E  
CS Sir William Dunn School of Pathology, Oxford University, UK.  
SO NUCLEIC ACIDS RESEARCH, (1992 Nov 11) 20 (21) 5655-60.  
Journal code: 0411011. ISSN: 0305-1048.  
CY ENGLAND: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)

LA English  
 FS Priority Journals  
 EM 199301  
 ED Entered STN: 19930129  
 Last Updated on STN: 19930129  
 Entered Medline: 19930104

AB We have investigated the developmental and tissue specific expression of the human embryonic zeta-globin gene in transgenic mice. A construct containing 550 bp of zeta-globin 5' flanking region, fused to a beta-galactosidase (lacZ) reporter gene and linked to the **locus control region** (LCR)-like alpha positive regulatory element (alpha PRE) was employed for the production of transgenic mice. Firstly, we compared the number of live born transgenic mice containing this construct to the number of live born transgenic mice containing the entire zeta-globin gene linked to the alpha PRE or the beta LCR. Data showed that 12% of mice generated from eggs injected with zeta-promoter/lacZ/alpha PRE DNA were transgenic compared to only 2% of mice generated from eggs injected with the entire zeta-globin gene linked to the alpha PRE or the beta LCR. The reduced number of live born transgenic mice containing the latter constructs suggests that death of transgenic embryos, possibly due to thalassaemia, may be occurring. X-gal staining of whole embryos containing the lacZ gene revealed that **zeta-globin promoter** activity was most pronounced at 8.5-9.5 days of development and was restricted to erythroid cells. By 15 days of development, no **zeta-globin promoter** activity was detected. These results suggest that the alpha PRE can direct high level expression from the **zeta-globin promoter** and that sequences required for the correct tissue and developmental specific expression of the human zeta-globin gene are present within 550 bp's of 5' flanking region. Sequences within the body of the zeta-globin gene or 3' of the cap site do not appear to be necessary for correct zeta-globin developmental regulation.

L3 ANSWER 82 OF 84 MEDLINE on STN  
 AN 92195871 MEDLINE  
 DN 92195871 PubMed ID: 1549512  
 TI LCR/MEL: a versatile system for high-level expression of heterologous proteins in erythroid cells.  
 AU Needham M; Gooding C; Hudson K; Antoniou M; Grosveld F; Hollis M  
 CS ICI Pharmaceuticals, Biotechnology Department, Macclesfield, Cheshire, UK.  
 SO NUCLEIC ACIDS RESEARCH, (1992 Mar 11) 20 (5) 997-1003.  
 Journal code: 0411011. ISSN: 0305-1048.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199204  
 ED Entered STN: 19920509  
 Last Updated on STN: 19970203  
 Entered Medline: 19920421

AB We have used the human globin **locus control region** (LCR) to assemble an expression system capable of high-level, integration position-independent expression of heterologous genes and cDNAs in murine erythroleukaemia (MEL) cells. The cDNAs are inserted between the human **beta-globin promoter** and the second intron of the human beta-globin gene, and this expression cassette is then placed downstream of the LCR and transfected into MEL cells. The cDNAs are expressed at levels similar to those of the murine beta-globin in the induced MEL cells. Heterologous genomic sequences can also be expressed at similar levels when linked to the LCR and **beta-globin promoter**. In addition we demonstrate that, after induction of differentiation, MEL cells are capable of secreting heterologous proteins over a prolonged time period, making this system suitable for use in continuous production systems such as hollow fibre bioreactors. The utility of the LCR/MEL cell system is demonstrated by

the expression of growth hormone at high levels (greater than 100 mg/l) 7 days after induction. Since the expression levels seen do not depend upon gene amplification and are independent of the integration position of the expression cassette, it is possible to obtain clones with stable high-level expression within 3-4 weeks after transfection.

L3 ANSWER 83 OF 84 MEDLINE on STN  
AN 92158607 MEDLINE  
DN 92158607 PubMed ID: 1741249  
TI The LCR-like alpha-globin positive regulatory element functions as an enhancer in transiently transfected cells during erythroid differentiation.  
AU Pondel M D; George M; Proudfoot N J  
CS Sir William Dunn School of Pathology, University of Oxford, UK.  
SO NUCLEIC ACIDS RESEARCH, (1992 Jan 25) 20 (2) 237-43.  
Journal code: 0411011. ISSN: 0305-1048.  
CY ENGLAND: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 199203  
ED Entered STN: 19920410  
Last Updated on STN: 19970203  
Entered Medline: 19920325  
AB A positive regulatory element (PRE) similar to the **locus control region** (LCR) of the human beta-globin gene cluster has recently been identified 40 kb upstream of the human zeta-globin mRNA cap site (Higgs D.R. W.G. Wood, A.P. Jarman, J. Sharpe, J. Lida, I.M. Pretorius, and H. Ayyub. 1990). We investigated the influence of the alpha PRE on human **alpha-globin promoter** activity in transiently transfected cells. The introduction of the alpha PRE into **alpha-globin promoter** /CAT expression constructs increased **alpha-globin promoter** activity by 15-30 fold in a human erythroid cell line (Putko) as well as in mouse erythroleukemia cells (MELCs) induced with hexamethylene bisacetamide (HMBA). When these constructs were introduced into uninduced MELCs or HeLa cells, only a 2-3 fold increase in **alpha-globin promoter** activity was observed. Deletion of 600 bp of alpha-globin 5' flanking sequences containing six putative SP1-binding sites had no significant effect on levels of **alpha-globin promoter** enhancement by the alpha PRE. We further demonstrated that the alpha PRE and HS2 of the beta-LCR could similarly enhance transcriptional activity of the SV40 early promoter in HMBA induced MELCs. Finally, we showed that **alpha-globin promoter** activity in the presence of the alpha PRE increased with continued HMBA exposure and was coincident with transcriptional activation of endogenous globin genes.

L3 ANSWER 84 OF 84 MEDLINE on STN  
AN 91230354 MEDLINE  
DN 91230354 PubMed ID: 1709381  
TI Functional properties of the beta-globin **locus control region** in K562 erythroleukemia cells.  
AU Moon A M; Ley T J  
CS Department of Medicine, Jewish Hospital, Washington University Medical Center, St Louis, MO 63110.  
NC DK 38682 (NIDDK)  
SO BLOOD, (1991 May 15) 77 (10) 2272-84.  
Journal code: 7603509. ISSN: 0006-4971.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Abridged Index Medicus Journals; Priority Journals  
EM 199106  
ED Entered STN: 19910707

Last Updated on STN: 19980206

Entered Medline: 19910619

AB In this report, we compare the function of the human beta-globin locus control region (LCR) in three K562 erythroleukemia cell assays, including (1) a transient transfection assay for "classical" enhancer activity, (2) a colony assay that detects "productive integration events," and (3) an assay that detects the ability of LCR fragments to confer hemin inducibility on linked, stably integrated gamma-globin promoters. Various LCR fragments were inserted into an expression vector consisting of an A gamma-globin promoter driving the neomycin phosphotransferase gene (gamma-neo). Using these vectors, we determined that a 2.5-kb DNA fragment containing LCR sites I through IV (previously named mu locus activation region [mu LAR]) had activity in all three assays; of the individual LCR sites, only site II was highly active in all three assays. One region within site II, consisting of tandem AP-1/NF-E2 consensus elements, had approximately 10% as much colony assay activity as the entire mu LAR. However, this region did not have detectable activity in a transient enhancer assay in uninduced K562 cells, nor was it capable of conferring hemin inducibility on linked gamma-globin promoters in stably transfected cells. Finally, we tested the ability of the mu LAR to activate promoters (beta-globin and cathepsin G) that are not normally expressed in K562 cells. beta-neo was minimally activated by the mu LAR in transient transfection experiments. The mu LAR increased the number of stably transfected colonies produced by beta-neo, but the absolute number of beta-neo colonies, with or without the mu LAR, was approximately 10% to 20% that of gamma-neo. In contrast, a minimal cathepsin G promoter was activated by the mu LAR in K562 cells. Our results suggest that LCR functions are dependent in part on the environments and the promoters with which the LCR is tested.

=> s beta globin promoter

470545 BETA

469 BETAS

470626 BETA

(BETA OR BETAS)

9966 GLOBIN

9769 GLOBINS

13089 GLOBIN

(GLOBIN OR GLOBINS)

97282 PROMOTER

22659 PROMOTERS

104410 PROMOTER

(PROMOTER OR PROMOTERS)

L4 185 BETA GLOBIN PROMOTER

(BETA(W) GLOBIN(W) PROMOTER)

=> d bib ab 180-185

L4 ANSWER 180 OF 185 MEDLINE on STN

AN 84041475 MEDLINE

DN 84041475 PubMed ID: 6314251

TI DNA sequence elements required for regulated expression of the HSV-1 glycoprotein D gene lie within 83 bp of the RNA capsites.

AU Everett R D

SO NUCLEIC ACIDS RESEARCH, (1983 Oct 11) 11 (19) 6647-66.

Journal code: 0411011. ISSN: 0305-1048.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 198312

ED Entered STN: 19900319

Last Updated on STN: 19970203

Entered Medline: 19831217

AB The genes of Herpes simplex virus type 1 (HSV-1) are classified into three temporally regulated groups. The Immediate-Early (IE) genes are transcribed first by the pre-existing transcription apparatus of the cell. The Early genes are transcribed only after IE-gene expression, and finally the Late genes are activated. The control of transcription of the HSV-1 glycoprotein D (gD) gene (an Early function) was studied by quantitative S1 mapping of RNA produced in HSV-1 infected HeLa cells after short-term transfection experiments using plasmids containing the gD promoter linked to the rabbit beta-globin gene. The viral promoter in the plasmid was activated in the same way as that in the virus itself; the RNA showed a similar time-course of appearance, dependence on prior IE-gene expression and pattern of RNA cap-sites. Deletion analysis showed that the DNA sequences necessary for Early promoter activation lie within 83 bp of the RNA cap-sites in this instance. Surprisingly, a plasmid-borne **beta-globin promoter** was also activated by HSV-1 infection. The mechanism of this activation, and DNA sequence similarities between the promoters of HSV-1 Early and rabbit beta-globin genes are discussed.

L4 ANSWER 181 OF 185 MEDLINE on STN

AN 84026511 MEDLINE

DN 84026511 PubMed ID: 6313220

TI Transcriptional activation of cloned human beta-globin genes by viral immediate-early gene products.

AU Green M R; Treisman R; Maniatis T

SO CELL, (1983 Nov) 35 (1) 137-48.

Journal code: 0413066. ISSN: 0092-8674.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 198312

ED Entered STN: 19900319

Last Updated on STN: 19970203

Entered Medline: 19831221

AB When the human beta-globin gene is transfected into Hela cells, no beta-globin RNA is detected unless the gene is linked to a viral transcription enhancer. In this paper we show that trans-acting adenovirus and herpesvirus (pseudorabies) transcriptional regulatory proteins can circumvent this enhancer requirement for detectable beta-globin transcription in transient expression assays. The viral gene products can be provided by constitutively expressed, integrated viral genes in established cell lines, by viral infection of permissive cells, or by transfection of cells with bacterial plasmids carrying the viral immediate-early genes. These results demonstrate the utility of transient expression assays for studying regulatory mechanisms involving trans-acting factors. Analysis of **beta-globin promoter** mutants indicates that between 75 and 128 bp of sequence 5' to the mRNA cap site is required for enhancer-dependent transcription in Hela cells. In contrast, beta-globin transcription in the presence of viral immediate-early gene products requires only 36 bp of 5'-flanking sequence, which includes the TATA box. Thus both cis and trans-acting viral factors activate beta-globin gene transcription in transient expression experiments, but the mechanisms by which they act appear to be fundamentally different.

L4 ANSWER 182 OF 185 MEDLINE on STN

AN 84026491 MEDLINE

DN 84026491 PubMed ID: 6194893

TI Human **beta-globin promoter** and coding sequences transcribed by RNA polymerase III.

AU Carlson D P; Ross J

SO CELL, (1983 Oct) 34 (3) 857-64.

Journal code: 0413066. ISSN: 0092-8674.

CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 OS GENBANK-J00093; GENBANK-J00094; GENBANK-J00096; GENBANK-J00158;  
 GENBANK-J00159; GENBANK-J00160; GENBANK-J00161; GENBANK-J00162;  
 GENBANK-J00163; GENBANK-J00164; GENBANK-J00165; GENBANK-J00166;  
 GENBANK-J00167; GENBANK-J00168; GENBANK-J00169; GENBANK-J00170;  
 GENBANK-J00171; GENBANK-J00172; GENBANK-J00173; GENBANK-J00174;  
 GENBANK-J00175; GENBANK-J00177; GENBANK-J00178; GENBANK-J00179;  
 GENBANK-K01239; GENBANK-K01890; GENBANK-K02544; GENBANK-M18047;  
 GENBANK-M19067; GENBANK-X00423; +  
 EM 198312  
 ED Entered STN: 19900319  
 Last Updated on STN: 19980206  
 Entered Medline: 19831217  
 AB We have investigated low abundance RNAs transcribed in vitro and in vivo from the human beta-globin gene. These RNAs contain globin mRNA sequences covalently linked to sequences transcribed from the 5' flanking region between -235 and the mRNA cap site (+1). Their synthesis in vitro is sensitive to high (100 micrograms/ml) levels of alpha-amanitin but not to low (2 micrograms/ml) levels, and one region of the DNA template bordering their 5' termini is similar to a small segment of Alu repetitive DNA and to the RNA polymerase III promoter consensus sequence. Therefore, these RNAs are transcribed by RNA polymerase III but extend into the mRNA-coding region that is usually transcribed by polymerase II. The polymerase III transcripts are polyadenylated and are probably spliced. Their presence in bone marrow cells and peripheral blood reticulocytes implies that they play some role in the erythroid cell.

L4 ANSWER 183 OF 185 MEDLINE on STN  
 AN 83297294 MEDLINE  
 DN 83297294 PubMed ID: 6310369  
 TI Differential activation of the mouse **beta-globin promoter** by enhancers.  
 AU Berg P E; Yu J K; Popovic Z; Schumperli D; Johansen H; Rosenberg M; Anderson W F  
 SO MOLECULAR AND CELLULAR BIOLOGY, (1983 Jul) 3 (7) 1246-54.  
 Journal code: 8109087. ISSN: 0270-7306.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 198310  
 ED Entered STN: 19900319  
 Last Updated on STN: 19970203  
 Entered Medline: 19831008  
 AB A series of plasmids was constructed to study the effect of two enhancers, the simian virus 40 72-base-pair repeat and the Harvey sarcoma virus 73-base-pair repeat, on the mouse beta maj-globin promoter. These plasmids contain the mouse beta maj-globin promoter linked to the Escherichia coli galK gene, thus allowing galactokinase enzyme activity to be used as a measure of promoter function. In CV-1 (primate) cells, it was found that an enhancer is required for optimal promoter activity and that the simian virus 40 (primate) enhancer increases galactokinase fourfold more than the Harvey sarcoma virus (mouse) enhancer. In L (mouse) cells, however, the Harvey sarcoma virus enhancer is 1.3-fold stronger than the simian virus 40 enhancer. These data support the hypothesis that enhancer activity can be species specific. Furthermore, when both enhancers are present on the same plasmid, their effect is additive on the **beta-globin promoter** whether the plasmid is in CV-1 cells or L cells.

L4 ANSWER 184 OF 185 MEDLINE on STN  
 AN 83155644 MEDLINE



DN 83155644 PubMed ID: 6187469  
 TI Isolation of transcription factors that discriminate between different promoters recognized by RNA polymerase II.  
 AU Dynan W S; Tjian R  
 SO CELL, (1983 Mar) 32 (3) 669-80.  
 Journal code: 0413066. ISSN: 0092-8674.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 198305  
 ED Entered STN: 19900318  
 Last Updated on STN: 19980206  
 Entered Medline: 19830527  
 AB A new fractionation procedure separates a whole-cell HeLa extract into three components required for accurate in vitro transcription. One component (Sp1) is a promoter-specific factor required for transcription of the SV40 early and late promoters, but not for transcription of other promoters we have tested. The second component (Sp2) is a general factor required for transcription of the SV40 promoters and a series of others, including the adeno-virus 2 major late promoter, the human **beta-globin promoter** and the avian sarcoma virus promoter. The third component is a fraction containing the endogenous RNA polymerase II. When SV40 and adenovirus templates were present simultaneously in an in vitro transcription reaction, addition of the Sp1 factor stimulated SV40 early promoter transcription 40-fold, and inhibited adenovirus major late promoter transcription by 40%. This finding suggests that Sp1 is involved in promoter selection, and is not merely a general transcription stimulatory factor.

L4 ANSWER 185 OF 185 MEDLINE on STN  
 AN 82060190 MEDLINE  
 DN 82060190 PubMed ID: 6946453  
 TI Transformation of frog embryos with a rabbit beta-globin gene.  
 AU Rusconi S; Schaffner W  
 SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1981 Aug) 78 (8) 5051-5.  
 Journal code: 7505876. ISSN: 0027-8424.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 198201  
 ED Entered STN: 19900316  
 Last Updated on STN: 19900316  
 Entered Medline: 19820120  
 AB In order to study the fate and possible expression of foreign DNA during embryogenesis of the frog *Xenopus laevis*, we have injected a rabbit beta-globin gene into fertilized *Xenopus* eggs. Frog embryo DNA was extracted at various stages of development, fractionated by agarose gel electrophoresis, transferred to nitrocellulose filters, and hybridized to labeled beta-globin recombinant plasmid DNA. It was found that the injected DNA replicated extrachromosomally, reaching, at gastrula stage, a level equivalent to a 10- to 200-fold amplification of input DNA. At later stages, a majority of the foreign DNA was degraded, but a small fraction was maintained. Six-week-old tadpoles as well as six-month-old frogs contained an average of 3-10 copies of the rabbit globin gene per cell. Most of these persisting globin genes were present as long tandem repeats and comigrated in agarose gel electrophoresis with high molecular weight *Xenopus* DNA. Analysis of globin gene expression by S1 nuclease mapping showed that the rabbit **beta-globin promoter** was recognized in the frog embryo and that the transcripts were correctly spliced.

=> d his full

(FILE 'HOME' ENTERED AT 13:35:03 ON 04 FEB 2004)

FILE 'MEDLINE' ENTERED AT 13:35:09 ON 04 FEB 2004

L1 387 SEA PLU=ON GLOBIN PROMOTER  
L2 84 SEA PLU=ON L1 AND LOCUS CONTROL REGION  
L3 84 SEA PLU=ON L1 AND LOCUS CONTROL REGION  
D BIB AB 80-84  
L4 185 SEA PLU=ON BETA GLOBIN PROMOTER  
D BIB AB 180-185

FILE HOME

FILE MEDLINE

FILE LAST UPDATED: 3 FEB 2004 (20040203/UP). FILE COVERS 1958 TO DATE.

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MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2004 vocabulary. See <http://www.nlm.nih.gov/mesh/> and [http://www.nlm.nih.gov/pubs/yechnbull/nd03/nd03\\_mesh.html](http://www.nlm.nih.gov/pubs/yechnbull/nd03/nd03_mesh.html) for a description on changes.

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COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

7.74

7.95

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